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0:00:05.6 Sarah Crespi: This is the Science Podcast for November 26th, 2021. I'm Sarah Crespi. Each week we bring you news and research published in science and the sister journals.

Have you noticed anything unusual about the trees lately? Maybe they seem a little extra nutty? Turns out on the East Coast of the United States, it's a masting year, when trees make more nuts, seeds, pinecones. Everything they drop, they're dropping it. Liz Pennisi is a staff writer for Science. In our first segment, we talk about the mystery of masting years.

Next, producer Meagan Cantwell talks with researcher Jean-Laurent Casanova about why some people are more vulnerable to severe diseases from viral infections. We're talking one mutation in the immune system could lead you to have a totally different reaction to a virus than somebody else.

Finally, in the newest installment of our series of books on race and science, books host Angela Saini talks with author Beverly Daniel Tatum about her seminal 2003 book, Why Are All the Black Kids Sitting Together in the Cafeteria?

Now we have Liz Pennisi. She's a staff writer for Science. Liz, what brought this story to your attention? How did you first hear about what's going on here?

0:01:26.0 Liz Pennisi: Living on the East Coast, I have been walking on acorns all fall. And they are dropping like crazy on the tops of my cars, on the roofs of my house. And so I had been thinking about them. And then the Physiological Transactions of the Royal Society came out with a special issue on masting. And I thought, "Okay, this is a sign, I need to do a story."

0:01:53.0 SC: Yeah. I've told a few people that it's a masting year, that that means that acorns are overproducing, other trees are making a lot of seeds. And they just... The light goes on, and they say, "Oh, that's why I'm getting acorns on my roof," or, "The squirrels are extra busy." And it's really been interesting to see it in action.

0:02:13.0 LP: Right. Exactly.

0:02:14.0 SC: We talk about acorns a lot for this year, are there any other trees that are going a little wild on the East Coast right now?

0:02:21.8 LP: Researchers have documented that spruces and pines and balsam fir are overproducing their cones this year.

0:02:31.1 SC: Okay.

0:02:32.0 LP: Yeah. So there's a lot of things producing a lot right now.

0:02:36.0 SC: We're gonna mostly talk about the East Coast of the US 'cause that's where it's

happening right now, but could this happen in other places too?

0:02:42.7 LP: So yes, you can have masting years throughout a continent. This happens in Europe a lot. And so what they mean by masting is, a whole bunch of trees, either of one species or of multiple species, decide to flower and produce seeds and nuts at the exact same time or close to the exact same time. And there are many ideas about why they do it some years and not others.

0:03:13.9 SC: So it's a regional coordination, and the regions can get pretty big.

0:03:17.8 LP: Yes. Yes.

0:03:19.3 SC: One thing we should talk about too is the downstream effects of a masting year. Who's eating seeds. Who's eating the seed eaters. Are there other by-products of a masting year that we should consider?

0:03:30.9 LP: Oh yes, certainly. One reason researchers think that masting occurs is so that when they have mast years and they produce a lot of fruit and nuts, whatever, there's more fruit and nuts on the ground than the squirrels can eat or the birds can eat. So there's plenty left over to germinate the next year. And then the reason that it's good to not do a mast year every year is, one, it takes a lot of energy for a tree to like produce so many acorns and so it doesn't have that energy every year. But two, if a forest produces a lot of nuts and seeds one year and produces very few the next few years, it means that all the rodents that have been feasting on those nuts won't have food for the following two years, and so their populations get smaller. And so then when they have a mast year, then there's plenty more seeds to be left over to sprout.

0:04:35.3 SC: Wow. So it's a long con against their rodent predators.

0:04:38.8 LP: Exactly.

0:04:40.4 SC: But this doesn't just stop at these small animals that eat the seeds. Then you can look at what eats them, and even further out, there are actually public health effects to mast years.

0:04:50.2 LP: That's right. So in terms of the public health effects, what they have documented both in the US and in Europe is that when you have a masting year, the next year you have a lot of mice. And the next year following, you have a lot of the ticks that carry Lyme disease because the ticks use mice as a host. So if there's a lot of mice, there's a lot of ticks that can thrive and then later on infect us. But you also have a situation where, because there's now a lot of mice and chipmunks and squirrels, the foxes and the lynxes and the owls and even the snakes that eat those animals can also increase in number because there's so much food around.

0:05:38.0 SC: There's a lot of ripple effects. It's not just the trees for one year. There's like two or three or four years of consequences on the trees, but also what eats the trees and what eats those things. It's really a massive phenomenon.

0:05:53.0 LP: Exactly.

0:05:53.9 SC: Well, next to this question of why it might happen, we talked about starving out their predators every couple of years, there's also a related question about the timing. Why are certain years masting years? A lot of theories link this to climate. The one I like here is this one where the trees are almost like predicting the weather in the future and setting out a lot of seeds ahead of that. How do they know what the weather is or what kind of weather might set up trees for a masting year?

0:06:24.5 LP: So, they don't actually know what the weather is. But weather can follow certain patterns, and that's because you have these major climatic events. These are called oscillations, and there's two that are important. One is called the North Atlantic Oscillation, in which the low-pressure and high-pressure areas at sea level flip-flop between the United States, North America, and Europe. And when they flip-flop, they bring different kinds of weather patterns. And so a weather pattern that might be good for encouraging a lot of seeds, say like a wet, warm winter, also later on creates a situation that would be beneficial for those seeds sprouting.

0:07:15.5 SC: It's not just, "Oh lots of nutrients are around today. Let's drop seeds." It's, "In the next season, when the seeds that we're dropping now are going to germinate, the weather is gonna be fortuitous."

0:07:26.3 LP: Correct.

0:07:27.5 SC: And what was the other oscillation that you were mentioning?

0:07:31.0 LP: The other one is called the El Niño Southern Oscillation, and that affects basically things that are around the Pacific. So Southeast Asia, the west coast of the United States, and you have interactions between these two oscillations that can create yet a different set of weather patterns.

0:07:50.6 SC: What about fires? That's got mentioned a few times in the story, that fire actually might be involved in masting years as well.

0:08:00.0 LP: Yes, that's right. So what can happen is, if you have the El Niño Southern Oscillation pattern create dry weather, that dry weather can lead to forest fires, and forest fires can be good for seedlings because it takes out all the competition, opens up that ground for young seedlings to sprout.

0:08:19.5 SC: So again, with really big cycles happening here, why has this been so hard to figure out? We know about these weather patterns, we know about these climate events, and we know... We often notice when the trees are going through a masting year. Why has it been so difficult to figure out the relationships?

0:08:36.3 LP: So, it's been hard to figure out the relationships because number one, the ecologists and the plant biologists that study masting don't really know a lot about climate oscillations, and people who study climate oscillations don't really know much about biology. The second problem

is, there hasn't been a compilation of data so that you can see, "Okay, well, we had masting events all over the western United States in this particular year." So there's no pattern that you can really discern.

0:09:11.8 SC: But that is on the horizon. There is a database that's coming around that's gonna help researchers answer some of these questions?

0:09:18.2 LP: Yes, that's right. Researchers in Europe started out by just compiling records of beach and Norway Spruce masting events throughout the last 200 years, and once they had all those events mapped out, they could then overlay when the climate oscillations were changing and sort of see a pattern. And now there is even bigger database that has more records in it from more places, and it includes a lot of species.

0:09:53.9 SC: If these databases can help pick out some of the important correlations for climate, these oscillations, maybe some other factors that people haven't even considered yet, and masting years, what will that mean when we get a better handle on this, can we predict masting years? What can we learn once that correlation's in place?

0:10:14.7 LP: Once we understand what triggers masting, we can have a better sense of when masting will occur. Once we have that sense, then we can have a better sense of how to prepare for the after-effects of masting?

0:10:26.3 SC: Like the bad stuff, all the ticks or overabundance of stoats?

0:10:32.7 LP: Yeah, so in New Zealand, there's a good example of this where conservationists use temperature differences from one season to the next to predict masting in the southern beach that covers the South Island. When the beaches mast, the number of mice increase and the number of their predator... Stoat increases as well. But of course, the masting lasts one season and then the mice have nothing to eat, so they decrease in number, and so the stoats then start turning to native birds for food. And conservationists worry about that and need to take action about that. And so if they can predict a masting year, they can then predict when they're gonna have to take countermeasures to control the stoat population down the road.

0:11:24.8 SC: What about positive effects of understanding the masting years? Can we take advantage of the fact that we're gonna have a bumper crop of acorns in some way?

0:11:33.8 LP: Well, if you're in Europe and you raise your pigs in the old-fashioned way, then what you do is you take advantage of these bumper crops and let your pigs run wild in the forest to fatten up.

0:11:47.3 SC: Liz, were you surprised that this was an unresolved question, that masting was pretty mysterious?

0:11:55.7 LP: I'm not surprised because there's so many factors that can go into whether or not a tree will produce a lot of seeds, produce no seeds, produce a small amount of seeds, that I could

imagine that it would be very hard to tease out what the actual trigger is and why that trigger exists.

0:12:15.7 SC: Thanks, Liz.

0:12:17.4 LP: Well, thank you. It's a very interesting topic.

0:12:20.9 SC: Yeah, Liz Pennisi is a staff writer for Science, you can find a link to the story we discussed at science.org/podcast.

Don't touch that dial, up next, producer Meagan Cantwell talks with researcher Jean-Laurent Casanova about what the genetics of the immune system can reveal about vulnerability to viral infection.

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0:12:47.4 Meagan Cantwell: There are over a quadrillion, quadrillion viruses that exist, yet only about a dozen can cause severe disease and inflammation in more than 1% of humans. This week in Science, researchers explore the varied roles that inflammation plays in our body, from metabolism, all the way to infection. I'm here with Jean-Laurent Casanova to discuss one of these angles, how genetics impact, who is more vulnerable to severe disease and inflammation? Thanks so much for joining me.

0:13:19.3 Jean-Laurent Casanova: Thank you for inviting me.

0:13:20.5 MC: Of course. It's surprising how often we're infected by viruses and don't even know it, why is it that some people exhibit symptoms from viral infections while others don't have any?

0:13:32.6 JC: Yes, most viruses are benign in most people, but still a number of viruses are life-threatening in one in 10,000, one in 100,000 for many, and 10% or more for others, but the question is the same, even though the proportions vary, why would a virus be life-threatening in a proportion of patients, and benign in others? It's what we like to call the infection enigma. Little is known, but for some viral infections, it's been shown over the last 20, 25 years that there are single gene inborn errors of immunity underlying specific viral disease, and that genetic defect is silent until the person is infected with the particular virus to which he or she is predisposed.

0:14:29.3 MC: Does an inborn error of immunity mean that an individual is more likely to get severe disease from just a singular virus or a whole host of different viruses?

0:14:40.0 JC: In a given individual, these inborn errors of immunity predispose the carrier to one viral disease, but the same genetic disorder in other people can occasionally underlie another viral disease.

0:15:00.3 MC: When you see inflammation as a side-effect to these severe diseases, what does that tip you off about? What does that tell you about what's happening in the body?

0:15:07.6 JC: What inflammation attests is to the fact that the steady state mechanisms of immunity against the invading virus have failed. If this first line of steady-state immunity fails, then there's a recruitment of additional leukocytes and their ensuing activation, and that defines inflammation. So inflammation attests to the failure of steady state immunity.

0:15:36.9 MC: With these single-gene inborn errors of immunity, are some of them linked to not producing enough of these leukocytes, these white blood cells that are defending the body?

0:15:46.9 JC: Well, interestingly no, the inborn errors of immunity underlying specific viral diseases do not really operate at the level of inflammation, they operate at the level of steady-state leukocytic, or more commonly, cell intrinsic immunity to viruses, which is really the very first line of defense, the virus enters the body and penetrates cells that express the ad hoc receptor, and in these cells, the virus replicates in pulmonary epithelial cells, for example, in that case, you develop viral pneumonia; or in neurons, in the brain, in that case, you develop viral encephalitis; or in keratinocytes in the skin, and in that case, you develop warts.

0:16:39.5 MC: You talk about a whole host of different examples of this in your review, and one of them that I thought was interesting was how one of these inborn errors, it causes something called Treeman syndrome. And that's a rare disease that usually results in lesions on the skin to be pretty debilitating as it builds on the skin, and it's interesting that it comes from a response to HPV, which usually a lot of people don't have symptoms for. So I'm curious if you could talk more about exactly how that manifests in the body?

0:17:11.9 JC: Well, that's a good example that you take because it's actually an exception or among the exceptions that inborn error of immunity underlying the Treeman syndrome does not paradoxically impact keratinocytes where the papillomaviruses replicate, the genetic defect impacts leukocytes specifically T-cells. These patients have a deficiency of CD28, which is a co-activator of T-cells, because T-cells circulate through the entire body, so why would the deficiency of CD28 selectively underlie the Treeman syndrome? That was a big surprise. So clearly, there are inborn errors of immunity that disrupts cell-intrinsic immunity, including in keratinocytes, but there are also inborn error of immunity that affect leukocytes, and they can underlie paradoxically a very narrow range of viral illnesses.

0:18:15.6 MC: Just because it is impacting these leukocytes, these white blood cells, it doesn't necessarily mean they're more vulnerable to all diseases in general, it's interesting that it's more targeting and specifically causing this syndrome?

0:18:28.6 JC: Yes, that's exactly right.

0:18:29.9 MC: So another example that follows more closely the typical relationship between these genetic mutations and cells is HSV encephalitis, usually people who are infected with the herpes simplex virus either have no symptoms or they have cold sore, something pretty minor, but in some children with HSV, they start to have massive inflammation in the brain, why is it that this is usually seen in children and not adults?

0:18:58.9 JC: The reason for that is that the primary infection, that is, the first time you're infected with this virus is in childhood. The same applies to papillomaviruses that infect keratinocytes and can cause warts. However, papillomaviruses are slow-growing, slowly-replicating viruses when compared with herpes simplex virus, and that accounts for herpes simplex encephalitis being an acute and even hyper-acute condition when compared with the Treeman syndrome that we discussed earlier because warts take much more time to develop and grow.

0:19:38.3 MC: So we've talked about disease that manifests on the skin and in the brain, and the last example in your review is talking about respiratory diseases. And I thought that covid-19 would be a pretty appropriate one to talk about. What are the genetic reasons that underlie why some people get really severe cases of covid-19, and others don't?

0:20:01.5 MC: The epidemiologists, early on during the pandemic, in the spring of 2020, found that the mortality of covid is strongly influenced by age. The risk of death, and by inference, the risk of critical or severe disease doubles every five years from childhood onward. So the question that we posed is, what are the molecular and cellular determinants of critical covid that would be consistent with this age-dependent pattern? The answer is that there are both inborn errors of Type I interferon immunity underlying critical covid-19; and auto-antibodies that are pre-existing and that neutralize Type 1 interferons.

0:20:54.0 MC: How does a deficiency in Type I interferons or an increase in autoantibodies cause more severe cases of covid 19?

0:21:02.4 JC: Type I interferons form a family of 17 molecules that bind the same receptor. One or another of these 17 interferons are produced by every known discernible cell type in the human body. They're produced upon viral infection, and their job is to stimulate cells nearby. It's a signal that tells cells near an infected cell, that a virus has infected a cell. And therefore the other cells are warned, and they're gonna shut down a number of programs to make them to some extent resistant to the viruses that may hit them in the waves that follow the first infection of a cell and the replication of the virus, and therefore the spread of new viruses. That's what Type I interferons do. Some patients have mutations from birth, inborn errors of immunity, that disrupt the production of these Type I interferons or their activity. And other patients have autoantibodies that bind to these interferons, and thereby neutralize their activity.

0:22:28.5 MC: So with these two mechanisms, there's a difference in the prevalence of each depending on age. Is that correct?

0:22:34.8 JC: The inborn errors of immunity are preferentially found in patients younger than 50 or 60 years, whereas the auto-antibodies to Type I interferons are preferentially found in patients older than 50, 60, or 70 years. The distribution of these auto-antibodies in the general population in uninfected individuals is relatively stable until age 60, 65, around 0.5%, and then there's a sudden, sharp rise in their prevalence, reaching 7% at age 80 years.

0:23:15.4 MC: Once you've dialed in on the mechanism that's causing severe disease, how does this guide therapeutics and potential treatments for a patient?

0:23:24.8 JC: Yes, if your inborn error impairs the production of Type I interferon, you can prevent disease or treat disease with either Type I interferon available commercially. In contrast, if you have an inborn error of immunity that prevents the activity of Type I interferon, it's not useful to use a Type I interferon, obviously. As for the auto-antibodies that neutralize interferons, they typically neutralize alpha and omega interferon, and only rarely beta interferon, which suggests that beta interferon can be used if given early in the course of disease. In both cases, I think what's really important is, A, to vaccinate them, and B, if infected and unvaccinated or if sick despite vaccination, then they would benefit from monoclonal antibodies neutralizing the virus.

0:24:21.2 MC: For people who have these inborn errors of immunity, getting vaccinated to something like HPV or to covid-19, is that still going to help them?

0:24:31.3 JC: Yes, absolutely. We don't have direct evidence that it does, but there's indirect evidence that it is the case because collectively these inborn errors and autoantibodies already account for almost 20% of critical cases. And we know epidemiologically that the vaccines, Pfizer vaccine or the Moderna vaccine, that they're efficient at 90%-95%, so by inference, they likely protect at least some patients with autoantibodies or inborn errors.

0:25:03.2 MC: It seems like researchers have uncovered a lot of different inborn errors of immunity tied to severe cases of disease. What is the next step in this field? Are there more to uncover, more viruses to better understand?

0:25:16.7 JC: So my lab will try to stay focused on these three infections we've discussed. We're gonna try to identify more genetic lesions and better understand the mechanism of disease at the molecular and cellular level. As for per the field, well, my hope, of course, is that other labs, other colleagues worldwide, try to better understand the molecular, cellular, and immunological determinants of the great many other viral illnesses that affect human beings.

0:25:44.5 MC: I'm looking forward to seeing that research. Thank you so much for taking the time to speak with me.

0:25:48.1 JC: Thank you very much.

0:25:49.4 MC: Jean-Laurent Casanova is the Levy Family Professor at the Rockefeller University, and an investigator at Howard Hughes Medical Institute. You can find a link to his review at sciencemag.org/podcasts.

0:26:03.4 SC: Stay tuned for the next installment in our series on books at the intersection of race and science. This month, host Angela Saini talks with psychologist Beverly Daniel Tatum about her classic, Why Are All the Black Kids Sitting Together in the Cafeteria?

0:26:23.9 Angela Saini: These days, talking about structural and systemic racism, unconscious bias, and the subtle ways in which racism shapes human behavior, feels normal. But before this, there was the work of psychologist, Beverly Daniel Tatum. I'm Angela Saini, science journalist and

host of this series of podcasts looking at books on science and race. We're up to episode five, and this month, I'm honored to be in conversation with someone who helped first bring to light exactly how pervasive and insidious the effects the racism are on everyday lives.

0:26:54.7 AS: Beverly Daniel Tatum's internationally best-selling book, Why Are All the Black Kids Sitting Together in the Cafeteria? Was published in 1997, and released in an updated edition in 2017. It set the framework for thinking about race in education. President Emerita of Spelman College, Tatum is an expert on the psychology of racism. At the heart of her work is identity, how we think about ourselves and of others, and how these ideas are formed from a remarkably young age.

0:27:26.0 AS: Beverly, thank you so much for joining me. What brought you to this subject of racism and education?

0:27:32.6 Beverly Daniel Tatum: I was born in 1954, which was an important year in race relations in the United States. It was the year of the Brown versus Board of Education's school board decision, which basically said, "Legalized school segregation is unconstitutional and illegal." And I often think it is significant that I was born that year.

0:27:54.3 BT: But I was born in September, that court decision was in May. I was born in Tallahassee, Florida, at a time when the South was still very much ruled by a system of segregation, "Jim Crow Segregation" as we called it. My parents were both college-educated at Howard University, and my father was a professor at Florida A&M, which is a historically black institution, teaching art.

0:28:19.6 BT: And he wanted to get his doctorate, he had a bachelor's degree and a master's degree, but wanted a doctorate so he could advance in higher education, and would have liked to do that in Florida. It would have been convenient. Certainly, he would have liked to do it at Florida State University, which was located just across town from the school where he was teaching.

0:28:38.0 BT: But unfortunately, even though the Brown versus Board of Education decision had been passed, the State of Florida, like a lot of Southern states in the US, did not immediately change their policies or their practices, and he was not able to attend Florida State because at that time, it was a whites-only institution.

0:28:57.4 BT: But what the State of Florida did to accommodate his right to education, was to pay his transportation out of the state. So he traveled from Florida to Pennsylvania and earned his doctorate at Penn State University. And I grew up in this small, malevolent town of Bridgewater, Massachusetts, where my dad was teaching at the university. My mother eventually became a school teacher in the town, and we were one of very few black families living there. At the time that I was growing up, I was often the only black child in my classroom. And so when I think about why I write about race, I think it has a lot to do with my growing up experience as an observer.

0:29:42.9 AS: Early on in your book, you described the conversation you had with your son when he was four. Your son does appear quite regularly in your work. And somebody told him at the age

of four that he was black, and he was confused because he looked at his skin and saw that it was brown. And you very beautifully told him that this was... Just because we call people black and white, that very rarely has very much to do with actual skin color, which varies obviously enormously within these categories. And it did make me wonder when I was reading how hard societies actually have to work to instill notions of social difference based on what can quite often be quite subtle, physical differences.

0:30:21.0 BT: That is quite true and we learn about these differences from a very early age. We notice, for example, that some people are being spoken to differently than other people, or treated differently, whether you see that on television or in the books that you're reading, or who is even included in the books that you're reading, whose pictures do you see, whose pictures are left out.

0:30:43.8 AS: And so young children starting from really toddlerhood are able to notice and comment on these differences and start to recognize what is sometimes referred to as "a racial hierarchy," recognizing that some people are seen as more valuable or more worthy or whose lives matter more than other people.

0:31:08.0 BT: And those assumptions get reinforced in ways that I think would probably horrify many parents if they gave it some thought. But in fact, it's really so much a part of everyday socialization. I sometimes refer to it, as I do in my book, as smog in the air. You don't see it, you don't think about it necessarily, but every day, you're breathing it in.

0:31:28.1 AS: And how hard was it for you to have these conversations with your children? 'Cause I know for me, it's been very, very difficult. How early should parents be starting?

0:31:37.4 BT: Well, I often say that children will let you know when they're ready to have these conversations because they'll start asking questions, as my son did. Some of those conversations took me by surprise, and I have to say it does help being a trained psychologist with a background in child development. So I wasn't completely flummoxed by some of what he had to ask about.

0:32:01.6 BT: But it's not uncommon for young children to, for example, point out physical differences. That white child who says to his or her mother, "Why is that person so dark?" Might get a "shh" in response, but that teaches, "This is not something we talk about. This is not something we comment about." A more appropriate or affirming or helpful response would be to simply say, "because people come in different shades just like flowers come in different colors, just like we have different hair color, some people have light hair, some people have dark hair, some people have blue eyes, some people have brown eyes, that's the diversity of the world. And isn't that a wonderful thing?"

0:32:45.0 BT: We can certainly get more specific, as I did with my son when someone said to him, "Your skin is brown because you drink chocolate milk." And he came home and asked me if it was true, and I said, "No, that's not true, your skin is brown because you have something in your skin called melanin, everybody has some, the more you have, the browner your skin is, it helps protect your skin from the sun. And it happens that at your school, you're the kid with the most, which is why your skin is browner than everybody else's. Everybody has some, doesn't have anything to do

with the milk you're drinking."

0:33:17.9 AS: There are, of course, the social and political implications that come with having a certain color of skin, so how do you broach that with children?

0:33:28.6 BT: As kids are learning these categories, and they do learn them, they're absorbing the information around them, we've been talking about how that information is absorbed, not always in a conscious way, but they're observing that some people live in certain neighborhoods, some people work in certain jobs, certain people speak with certain accents. All of this is part of what they're learning as they are trying to understand the way the world works. And when they start to learn the names of these groups, we have given different groups, different names, when children start to learn those names, they are also learning the values that are attached. We as adults used use words like racism, sexism, homophobia, but for a young child, the concept of fair and unfair is often most understandable. Even preschoolers get that some things are not fair, and if you can talk about what's fair and not fair, and what we do when things are unfair to try to correct that, that's a conversation that a preschooler can grasp very easily.

0:34:33.3 AS: One of your observations is that it's around the sixth or seventh grade that children start separating into racial groups in a way that they socialize, and this is presumably where your book title comes from. Why is this? Why does it happen around that age?

0:34:47.7 BT: When we think about racial identity, this is something that develops over time in terms of a child's understanding of what group they belong to, what the meaning of that group membership is, what the social meaning of it is in terms of status or stigma, etcetera. But when we think about why that happens in sixth or seventh grade, when you think about that in terms of adolescent development... So it's not just that you are now older, but your brain has changed. As you get older, your brain matures and you're able to grasp more abstract concepts. So the idea of thinking about, "Who am I? Where do I fit in the world?" These are adolescent questions. People respond differently to you when you look like a young woman than when you look like a little girl.

0:35:40.8 BT: People respond to you differently when you look like a young man who might be dangerous, if you've got stereotypes around criminality, it's that it's associated with darkness or dark skin than if you are a cute five-year-old. So the social responses that you're getting from other people are triggering you to think about, what does it mean to be a person who looks like me moving through the world? How do people respond to me? And if I'm thinking about those questions, I'm wondering who else is having this experience, who else knows what it's like to be followed around in the store by the security guard, or being perceived as potentially at risk for success or lack thereof, who else has been stereotyped in these ways? And it's a natural thing to connect with people who are having a shared experience.

0:36:35.0 AS: I'd like to bring all these learnings into the world of science, because one of the problems in science obviously is a demographic one. Black scientists we know from studies are less likely to be hired, funded, retained, or promoted even when they have the same qualifications. Most scientists I meet as a journalist, tell me that they're not racist, the problem isn't theirs, that it lies somewhere else. So how can we address this?

0:37:00.4 BT: Racism is not just about individual acts of meanness. If you think of yourself as a good, kind-hearted person, you will first say, "I'm not racist, I'm not calling people names, I'm not burning crosses on anybody's lawn, I'm not actively discriminating in ways that are obvious to me and other people." But what we know is that a lot of the bias that gets expressed isn't always conscious, it isn't always part of our conscious awareness, but we see the results of it, just the fact that it's not conscious doesn't mean it doesn't have an impact. So if I am more comfortable in an interview with someone who shares my background, then I may respond more warmly in that interview, I might ask more friendly questions, I may afterwards rate that person more highly because of the comfort level I have.

0:37:56.9 BT: And if we find routinely that there are people who are being left out, that suggests that there's something wrong with our process, not that there's something wrong with those scientists. And if we understand that there's something perhaps wrong with our process, we can shift our process in ways that reduce the bias in it. For example, we can recruit more aggressively in places where there are likely to be more diverse candidates. I served for many years as the president of Spelman College, which is the leading educator of black women who go on to earn PhD's in the sciences. If you want to have more black women in your science graduate programs, you ought to be recruiting as Spelman.

0:38:40.9 AS: One of the most valuable things about your book is that you turn around the lens, and this feels quite normal now in 2021, it was quite radical, I imagine, in 1997, was to turn around the lens onto whiteness, onto those people who don't think about race because they feel they don't need to. So what is it like for people to confront this idea of racial privilege or racial advantage, particularly someone like academia, which often considers itself to be meritocratic?

0:39:09.3 BT: Yes, I find that it's very difficult for people, people who have privilege to name it or recognize it, and it's understandable in this sense.

0:39:19.1 BT: If you are an able-bodied person walking down the street, you don't notice every curve cut, right? Because you don't need to. If there's a step, you step up. If there's a step, you step down. You're not thinking, "Gosh, if I was in a wheelchair, how would I navigate this?" Because you don't have to. You're not thinking about it. In the same way, people who are white and who receive the benefit of the doubt in a job interview, who receive the common courtesies in a shop from a shopkeeper, who drive home without worrying about being stopped by the police. When they get home at the end of the day, they don't say, "Gosh, my life was much easier today because of my whiteness." They don't think about that, and yet, if you were experiencing those things, if you were stopped by the police and had a negative interaction because you are a person of color, or if you had to stop at the grocery store and experience some microaggressions in your interaction with the shop keeper who was not as polite or gracious to you as the person in front of you in the line. If those things had happened to you, you'd come home and you'd be feeling worn out because of those experiences. The person who wasn't having them isn't thinking about what didn't happen that day.

0:40:38.8 BT: There is a scholar whose work I admire, who wrote a book called, White Guys on Campus, which is about young white men in university. He talks to them about privilege. The scholar's name is Nolen L. Cabrera. And he says, "I've come to use a different term, I don't call it privilege anymore. I call it immunity, racial immunity, because in some ways, it's not so much that

you're being given things, but that things aren't happening to you, you're immune to some of the bad things that other people of color are experiencing." That immunity makes life easier for you. It means you can focus on your studies, it means you can move through the world with lower blood pressure, it means that you can have a better day on average. It doesn't mean you're never gonna have challenges, and that's when I hear white people pushing back against the idea of privilege, they're often doing so because they say, "I struggle. I grew up poor, I struggle in my life. What do you mean I'm a "privileged person?" It doesn't mean all your problems have been solved. It does mean you're getting the benefit of the doubt a lot of the time.

0:41:46.8 AS: Wonderful, thank you so much, Beverly Tatum. It's been an honor speaking to you.

0:41:51.3 BT: It's been my pleasure, thank you so much.

0:41:52.1 AS: And thank you at home. Next month, we have our sixth and final episode featuring not an academic, but a science fiction writer, Tade Thompson. Exploring how we might imagine race in the future. I'm Angela Saini, and I hope you'll join me then.

0:42:08.4 SC: And that concludes this edition of the Science Podcast. If you have any comments or suggestions, write to us at sciencepodcast@aaas.org. You can listen to this show on the Science website at science.org/podcast. You can subscribe there, or anywhere you get your podcast. This show was edited and produced by Sarah Crespi, with production help from Podigy, Meagan Cantwell, and Joel Goldberg. Transcripts are by Scribie. Jeffrey Cook composed the music. On behalf of Science Magazine and its publisher, AAAS, thanks for joining us.